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ORIGINAL ARTICLE

Polychlorinated biphenyls (PCB), thyroid hormones and cytokines in construction workers removing old elastic sealants

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Abstract
Objectives To estimate the internal PCB level in Swedish workers specialised in PCB abatement in buildings and to measure possible effects of PCB on thyroid function and aspects of the immune system.
Methods Thirty six of 40 eligible workers (90%) removing old elastic sealants containing PCB and 33 control construction workers provided blood samples for determination of 19 PCB congeners and some other organochlorine compounds (hexachlorobenzene and p,p'-DDE), thyroid function hormones and a set of cytokines. The PCB exposed group was reinvestigated after 10 months for a trend assessment.
Results The sum of 19 PCB congeners in blood plasma from the occupationally PCB-exposed group was twice the level in the controls (geometric mean 580 vs. 260 ng/g lipid; $P < 0.001$), and there was also some difference in p,p'-DDE between the groups while the lipid-adjusted hexachlorobenzene levels were of the same magnitude. No statistically significant increase in overall PCB levels was observed in the abatement workers at follow-up and some congeners even declined. Thyroid function was not associated with PCB exposure at the current levels and this applied also to the cytokines investigated.
Conclusions Swedish workers removing old elastic sealants with PCB have a higher internal PCB load than unexposed colleague construction workers, tentatively secondary to historical exposure. A system of protective measures seemed to be efficient since no further increase was noted after a longish period of additional exposure. There was no evidence of thyroid function or immune system involvement, as expressed by a set of cytokines, at the low PCB levels recorded.

Keywords Polychlorinated biphenyls · PCB · Hexachlorobenzene · Environmental monitoring · Thyroid function tests · Cytokines

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Introduction

Beginning in the late 1950s polychlorinated biphenyls, PCB, were widely used in the Swedish building sector as plasticisers for elastic sealants but from 1973 the use of PCB in open applications, as an additive in sealants, was banned. Before the ban, approximately 100,000 metric tonnes of elastic sealants with variable amounts of PCB were introduced to apartment houses, offices and other buildings (Jansson et al. 1997). In 1998 a network within the Swedish building and real estate sector launched a national campaign to eliminate elastic sealants containing PCB from all buildings before 2003 (Ecocycle Council 1998). Similar initiatives were organised in some other Nordic countries.

As part of the Swedish campaign studies were carried out to find the most efficient and safe methods for PCB removal. In this process, exposure measurements showed high levels of PCB in the worker's vicinity during certain operations (Sundahl et al. 1999) and detailed safety instructions were launched by the appropriate sector of the domestic construction industry. Besides authorisation of contractors, essential components included disposable gloves and coveralls, self-contained respirators and hand-held cutting and grinding tools connected to portable dust extractors equipped with microfilters (Svenska Fogbranschens Riksförbund 1999).

The campaign, however, proved somewhat overambitious and by the end of 2001 only some 10% of the goal had been achieved (Swedish Environmental Protection Agency 2002). To obtain an estimate of the internal PCB exposure level so far in construction workers specialised in PCB abatement and to establish a basis for the future we adopted a cross-sectional as well as a prospective approach. As indicators of potential direct biological effects of PCB we included measurements of thyroid function and involvement of the immune system through cytokine analysis. As indicators of general background exposure to some other persistent organochlorine compounds (OCC), hexachlorobenzene and *p,p′*-DDE (1,1-dichloro-2,2-*bis*(*p*-chlorophenyl)ethylene), a major metabolite of the insecticide DDT (1,1,1-trichloro-2,2-*bis*(*p*-chlorophenyl)ethane), were included in the analytical protocol.

Material and methods

Subjects and procedures

Details of the target population and the identification of the study group have been described elsewhere (Wingfors et al. 2006). In short, after a nation-wide call to appropriate contractors 36 of 40 eligible male abatement workers (90%) with at least 6 months experience of PCB removal in the two previous years (2000–2001) agreed to participate in the study along with an age and sex-matched control group of construction workers without occupational PCB exposure ($n = 34$). The fieldwork was conducted in the spring 2002. Ten months later 28 subjects (78%) from the exposed group were available for a reassessment that included information on intervening PCB abatement.

All subjects completed a self-administered questionnaire covering aspects of their general health, tobacco habits and an occupational history including PCB contact, if any, as well as use of respirators in dusty work. Background PCB exposure was assessed from questions on consumption of fatty fish and residence in a house with elastic sealants possibly containing PCB.

In the morning of an ordinary working day, we collected 40–50 ml blood in heparinised BD Vacutainer vials (Becton-Dickinson, Plymouth, UK) for analysis of PCB and some other OCC. Participants were asked to refrain from a fatty morning meal but they were not fasting. After centrifugation, plasma was transferred to acid washed borosilicate glass vials (KIMAX, Kleinfeld Labortechnik, Gehrden, Germany). Plasma was also collected in BD K3-EDTA vials for quantification of a set of cytokines, whereas serum for quantification of thyroid hormones was collected in BD vials with a coagulation activator. Body mass index (BMI) was calculated from standardised measurements of height and weight.

All blood samples were stored at −80°C until analysis.

Chemical analyses

Again, details of the analytical procedures for the OCC are available elsewhere (Wingfors et al. 2006). Basically, we determined the levels of 19 PCB congeners, HCB and *p,p′*-DDE using a solid phase assisted liquid extraction technique, Chem-Elut and 2-propanol/hexane, followed by high resolution gas chromatography-mass spectrometry (Päpke et al. 1989, Wingfors et al. 2005). The levels of various OCC in the samples were recorded on a wet weight basis but also after adjustment to the total lipid content as determined gravimetrically.

PCB results were expressed individually for each congener and as the sum of 19 individual chlorinated biphenyls (Σ19 PCB) as well as sum of seven (Σ7 PCB). These seven PCB are congeners #28, 52, 101, 118, 153, 138, and 180.

For an assessment of time trends in PCB burden, the results of the current investigation were compared with a group of historical controls (male construction material industry and food industry workers; $n = 60$; median age 44 years, range 26–63 years) from a previous study (Seldén et al. 1997). The plasma samples for this study were collected in the autumn 1994 and the PCB analyses were conducted at the same laboratory with similar methods.

Serum levels of the thyroid hormones total triiodothyronine (S-T_3) and free thyroxine (S-FT_4) as well as the pituitary derived thyroid stimulating hormone (S-TSH; thyrotropin) were determined at the Department of Clinical Chemistry, Örebro University Hospital, with time-resolved fluoroimmuno assays (AutoDELFIA, Wallac Oy, Turku, Finland), whereas a batch of cytokines (IL-2, IL-4, IL-6, IL-10, TNF-α, IFN-γ) were determined (limits of quantification, LoQ, 2.6–7.1 pg/ml) with flow cytometry (Human Th1/Th2 Cytokine CBA Kit-II, BD Biosciences Pharmingen, San Diego, CA) at the Clinical Research Centre, Örebro University Hospital. According to the kit manufacturer, the coefficient of variation was 3–4% for repeated analyses at low levels of cytokines.

The Örebro County Council human research ethics committee approved the study (decision no. 93/02) and informed consent was obtained from all participants.

Statistical methods

Analytes below the LoQ were assigned values of half this level (Hornung and Reed 1990). Differences in plasma levels of PCB between groups were investigated with Student's *t*-test after logarithmic transformation and consequently geometric rather than arithmetic means were used. Longitudinal changes within the exposed group were analysed with *t*-test for paired observations. To reduce the influence of some extreme values of *p,p′*-DDE, the non-parametric Mann–Whitney *U*-test was also applied. The association between the two samples from the exposed group was estimated with Pearson's correlation (r) except *p,p′*-DDE, where Spearman's rho (r_s) was used.

Differences in PCB levels between contemporary exposed workers and controls and historical controls were tested with regression analysis, adjusting for age. The association between PCB and thyroid hormone status was analysed with r_s, since the distribution of thyrotropin in particular appeared to be neither normal nor log-normal (cf. Demers and Spencer 2003).

Due to low rates of cytokines above the LoQ those variables were treated as dichotomous in the analysis. Accordingly, differences between groups were evaluated with Fisher's exact test and change from baseline to follow up with the Sign test providing exact *P*-values based on the Binomial distribution. For dose-response relationship with ∑19 PCB (after log transformation) logistic regression was applied to baseline values for both abatement workers and controls.

Results

Basic characteristics of the study population are displayed in Table 1. One control subject reported previous occupational PCB contact and was excluded from the study and another control was unable to provide serum for analysis of thyroid hormones. The proportion of smokers was higher in the exposed group than among controls (39 vs. 9%; $P < 0.01$), but there were no significant differences between groups with regard to age, BMI, oral use of moist snuff (smokeless tobacco; Swedish "snus"), consumption of fatty fish or subjective perception of general health. Interestingly, almost all subjects considered their current work as dusty but eight exposed subjects (22%) denied using any respiratory protection in dusty work. This behaviour was even more pronounced among controls (82%). The questions on current or historical residence in a building with PCB-containing sealants were insensitive and some 70% of both groups chose a "do not know" response (not in table).

Table 1 Some background characteristics of the study population

Characteristic	Abatement workers $n = 36$	Controls $n = 33$
Age, years		
Range	24–57	20–46
Median	32	31
Body mass index kg/m^2		
Range	20.1–34.3	20.9–31.4
Median	26.0	24.9
Tobacco habits *n* (%)		
Non-smoker	17 (47)	27 (82)
Ex-smoker	5 (14)	3 (9)
Smoker	14 (39)	3 (9)
Moist snuff (smokeless tobacco) user	20 (56)	13 (39)
Fatty fish meals *n* (%)		
Rarely/never	32 (89)	27 (82)
1–2/month	4 (11)	6 (18)
≥1/week	0 (0)	0 (0)
Current work dusty *n* (%)		
No	1 (3)	0 (0)
Yes	35 (97)	33 (100)
Respiratory protection in dusty work *n* (%)		
None	8 (22)	27 (82)
Filter mask	4 (11)	6 (18)
Filter mask/self-contained respirator	6 (17)	0 (0)
Self-contained respirator	18 (50)	0 (0)
Self-assessed general health *n* (%)		
Good	34 (94)	30 (91)

PCB

The overall plasma PCB level expressed as Σ19 PCB or as Σ7 PCB, whether on a wet weight or lipid-adjusted basis, was approximately twice as high in the exposed group of abatement workers compared with the control group (Table 2). This difference between the groups was statistically highly significant ($P < 0.001$) and applied for all individual PCB congeners on a wet weight basis. For PCB congener #180, however, the difference between the groups was not significant comparing lipid-adjusted values. With no obvious relation to PCB exposure, statistical significance was also achieved for *p,p′*-DDE, whereas the difference between the groups with regard to HCB was significant for the wet weight based comparison only.

Table 2 Geometric mean and range of PCB, HCB and *p*, *p*' -DDE in plasma of PCB abatement workers ($n = 36$) and controls ($n = 33$) on a wet weight basis (ng/g) and after lipid adjustment (ng/g lipid)

Compound	Wet weight				Lipid-adjusted			
	Abatement workers		Controls		Abatement workers		Controls	
	GM	Range	GM	Range	GM	Range	GM	Range
PCB (IUPAC[a])								
28	0.052***	0.0029–0.39	0.011	0.002–0.032	13***	0.7–110	3.2	0.2–28
52	0.023***	0.001–0.20	0.0037	0.0009–0.037	5.5***	0.1–43	1.0	0.2–18
47	0.015***	0.0029–0.087	0.0036	0.001–0.012	3.7***	0.6–18	1.0	0.2–4.4
44	0.013***	<0.001–0.16	0.0010	0.00051–0.019	3.1***	<0.2–35	0.26	<0.1–9.3
74	0.096***	0.014–0.56	0.012	0.005–0.028	24***	3.0–160	3.5	1.5–11
70	0.0087***	<0.001–0.17	0.0014	0.00039–0.021	2.1***	<0.2–41	0.38	<0.1–10
66	0.065***	0.0041–0.76	0.0028	0.001–0.011	16***	1.1–220	0.8	0.2–2.7
56/60	0.036***	<0.001–0.40	0.0012	0.00089–0.0048	8.4***	<0.1–115	0.31	<0.1–1.5
95	0.028***	0.0018–0.19	0.0024	<0.001–0.011	6.9***	0.65–52	0.70	<0.3–2.7
101	0.038***	0.0044–0.32	0.0055	0.001–0.041	9.3***	0.8–70	1.6	0.3–20
99	0.053***	0.011–0.20	0.018	0.008–0.048	13***	3.4–59	5.2	2.9–15
87	0.010***	<0.001–0.076	0.0012	0.00075–0.0047	0.92***	0.27–16	0.30	<0.1–2.3
110	0.028***	0.0002–0.24	0.0025	0.00083–0.014	6.9***	0.54–52	0.71	0.23–5.4
118	0.11***	0.018–0.59	0.033	0.016–0.084	28***	5.2–170	9.4	4.2–64
105	0.034***	0.066–0.21	0.0061	0.0015–0.020	8.4***	1.7–61	1.8	0.5–10
153	0.51***	0.13–1.6	0.29	0.14–0.77	130**	37–540	84	31–360
138	0.46***	0.10–1.5	0.21	0.10–0.55	110***	29–640	59	25–200
182/187	0.086***	0.018–0.34	0.041	<0.003–0.12	16**	5.1–110	11.8	<2.1–92
180	0.35**	0.097–1.4	0.24	0.12–0.64	87	28–330	70	25–410
∑7 PCB	1.6***	0.40–4.9	0.80	0.40–2.0	410***	120–1800	230	90–1100
∑19 PCB	2.3***	0.56–7.8	0.90	0.45–2.2	580***	160–2200	260	110–1200
HCB	0.16*	0.067–0.32	0.13	0.063–0.24	39	19–98	37	16–140
p,p′-DDE	0.96***	0.31–12	0.54	0.23–1.8	240*	90–3100	160	35–410
Plasma lipids[b] (%)					0.42 (0.10)	0.23–0.64	0.37 (0.14)	0.12–0.73

PCB congeners sorted as they appeared chronologically in the chromatograms within each homologue group (tri to hepta)

[a] International Union of Applied and Pure Chemistry

[b] Arithmetic mean (standard deviation)

* $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$

Among occupationally exposed workers available for reassessment after 10 months ($n = 25$; three subjects without intervening PCB-related work excluded), the overall PCB burden (Σ19 PCB; lipid-adjusted) was practically unaltered in the follow-up plasma samples (Table 3). For some congeners, notably #52, 47, 44, 70, 95, 101, 87, and 110 significant reductions were found, but the contribution of these congeners to the overall measure was limited. The correlation between the samples was high for each compound and group of compounds (r range 0.58–0.94; median 0.88). Results were very similar using PCB values on a wet weight basis. Subjects reporting no use of respiratory protection ($n = 5$; three subjects from baseline missing) showed a GM Σ19 PCB increase of 12 ng/g over the observation period as opposed to the other workers ($n = 20$) who presented a slight decrease of -3 ng/g (not in table).

In the trend analysis, the GM Σ7 PCB (lipid-adjusted) concentrations in the controls of this study was 60% lower than in the historical controls of construction material industry and food industry workers (Table 4), suggesting a substantial reduction of general background PCB exposure over the period of less than eight years covered by the comparison. This difference was evident also after adjustment for age in the current and the historical controls. The group differences were homogenous over most congeners. Even in the occupationally exposed group of abatement workers, the GM Σ7 PCB level was lower than the historical controls although not statistically significant after age adjustment (410 vs. 580 ng/g lipid; $P = 0.08$). For HCB, however, no

Table 3 Levels of PCB, HCB and *p, p′*-DDE in baseline and follow-up samples (10 months later) of plasma from PCB abatement workers ($n = 25$). Geometric mean (GM), range of lipid-adjusted values (ng/g lipid) and correlation (Pearson) between the samples

Compound	Baseline		Follow-up		
	GM	Range	GM	Range	r[b]
PCB (IUPAC[a])					
28	18	2.9–110	16	2.4–87	0.90
52	7.7	0.91–43	4.0***	0.58–20	0.81
47	4.8	0.56–18	3.8*	0.63–15	0.84
44	4.0	<0.2–35	2.1**	0.13–12	0.66
74	30	3.0–160	33	3.4–150	0.94
70	2.8	<0.2–41	1.1***	0.28–4.4	0.58
66	25	1.2–220	25	1.2–170	0.91
56/60	12	<0.1–115	15	0.97–86	0.91
95	9.8	1.2–37	8.2*	0.80–38	0.91
101	14	2.5–62	10*	1.0–68	0.86
99	14	3.4–59	14	2.9–48	0.91
87	3.5	0.54–16	2.6**	0.35–8.8	0.88
110	9.5	1.5–52	6.3**	0.75–38	0.83
118	32	5.2–170	35	4.3–140	0.90
105	10	1.7–61	9.5	1.7–46	0.89
153	120	37–410	130	33–380	0.91
138	110	36–370	120	23–370	0.88
182/187	20	6.0–79	21	5.0–75	0.91
180	81	29–330	89	23–340	0.88
∑7 PCB	410	130–1400	430	87–1200	0.87
∑19 PCB	600	170–2300	610	110–1900	0.77
HCB	40	19–98	36	20–79	0.77
p,p′-DDE	240	100–3100	230	77–2900	0.89

[a] International Union of Pure and Applied Chemistry

[b] All correlations significant at $P < 0.001$ except PCB #70 at $P = 0.003$, Spearman's rho (r_s)

* $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$

significant change in concentration over time was noted, indicating a rather stable background exposure.

Thyroid hormones

All participants of the study had a normal thyroid function as measured by the hormones S-T_3, S-FT_4 and thyrotropin and there was no difference in hormone levels between occupationally PCB exposed abatement workers and controls (Table 5). In the occupationally exposed group observed twice over a 10-month period, the levels of thyroid hormones and thyrotropin remained stable (data not shown).

Combining occupationally exposed workers and controls ($n = 68$) to obtain a wider spectrum of PCB as independent variable, no statistically significant correlation with thyroid function parameters, positive or negative, was observed for either the individual congeners of Σ7 PCB, for Σ7 PCB or Σ19 PCB (Table 6).

Table 4 Levels of PCB and HCB in plasma from the controls of the current study and a group of historical controls of construction material industry and food industry workers (Seldén et al. 1997). Geometric means of lipid-adjusted values (ng/g lipid)

Compound	Current controls $n = 33$	Historical controls $n = 60$
PCB (IUPAC[a])		
28	3.2*	2.1
52	1.0	1.0
101	1.6***	3.0
118	9.4***	27
153	84***	230
138	59***	160
180	70***	140
∑7 PCB	230***	580
HCB	37	29

[a] International Union of Pure and Applied Chemistry

* $P < 0.05$, *** $P < 0.001$

Cytokines

The distribution of cytokine levels in plasma for the PCB-exposed abatement workers and controls is displayed in Table 7, which also shows that quantifiable values (≥LoQ) were obtained in a minority of cytokine determinations. This limitation precluded a more elaborate statistical analysis of the data, but there was no significant difference between the groups in the proportion of quantifiable cytokine values. Within the exposed group a significantly higher proportion of quantifiable values were observed in two of the six investigated cytokines at follow-up as compared to baseline (IL-10 $P = 0.035$, IFN-γ $P = 0.022$; remaining P-values > 0.18), but they did not relate to PCB levels in a dose-dependent way. The results from the logistic regression analyses showed that the odds for quantifiable cytokine levels decreased with approximately 30% for each logarithmic unit of ∑19 PCB, but the decrease was not statistically significant (P-values 0.27–0.44). The estimates were about the same for all cytokines (IL-2 and IL-4 not analysed). The similarity in results from the four regression analyses was due to a low intraindividual variation in cytokine levels, where 77% of the workers had all four values either below or above the LoQ (again IL-2 and IL-4 disregarded).

Discussion

In the cross-sectional approach, the PCB abatement workers had a higher PCB body burden than unexposed

Table 5 Serum levels of triiodothyronine (S-T_3), free thyroxine (S-FT_4) and thyrotropin (S-TSH) in PCB abatement workers and controls (arithmetic mean, AM, and standard deviation, SD)

Thyroid hormone	Reference values	Abatement workers $n = 36$		Controls $n = 32$	
		AM (SD)	Range	AM (SD)	Range
S-T_3 nmol/l	1.3–2.5	2.20 (0.26)	1.7–2.9	2.09 (0.26)	1.6–2.7
S-FT_4 pmol/l	9–20	14.2 (2.0)	10.8–18.6	13.9 (1.6)	11.9–19.1
S-TSH mU/l	0.5–4.2	1.68 (0.62)	0.22–2.9	1.71 (0.98)	0.5–5.3

Table 6 Spearman's correlation between PCB in plasma (ng/g lipid) and thyroid hormones S-T_3 and S-FT_4, and thyrotropin (S-TSH), for PCB abatement workers and controls combined ($n = 68$)

PCB # (IUPAC[a])	Hormone		
	S-T_3	S-FT_4	S-TSH
28	0.13	−0.01	−0.05
52	0.09	−0.13	0.03
101	0.15	−0.07	0.06
118	0.11	0.02	−0.04
153	0.01	−0.07	−0.04
138	0.07	−0.05	−0.03
180	−0.09	−0.08	−0.13
Σ7 PCB	0.04	−0.04	−0.07
Σ19 PCB	0.07	0.00	−0.11

[a] International Union of Pure and Applied Chemistry

colleague construction workers without occupational PCB exposure, indicating a differential PCB exposure between the groups probably explained by less stringent protection of the exposed group prior to the implementation of the current safety regulations. Prospectively, however, no evidence of additional PCB uptake was observed on a group level over 10 months of additional exposure for the abatement workers, suggesting an efficient worker's protection programme and good personal hygiene. Additionally, the significant reduction of some congeners (#52, 47, 44, 70, 95, 101, 87, and 110) indicated that elimination exceeded uptake during the study period since these congeners have low relative human accumulation factors (Brown 1994) and are regarded as more rapidly metabolised and excreted, as noted also in our previous work (Wingfors et al. 2006).

Johansson et al (2003) found elevated levels of low-chlorinated PCB (notably #28 but also #74, 66 and 99) in residents of buildings with PCB-containing elastic sealants as compared to controls from buildings without such sealants. The median PCB concentration in the control group of that study was well in accordance with what is reported here from non-occupationally exposed (control) workers, suggesting a good external validity, allowing for some methodological differences, in terms of domestic background PCB blood levels from the early 2000s.

The more long-term trend analysis showed evidence of substantially reduced background PCB levels in Swedish male workers between the mid 1990s and early 2000s. These findings are congruent with and corroborated by several longitudinal studies of breast milk from Swedish women and blood from male Swedish conscripts as well as older men (Norén and Meyronité 2000; Lignell et al. 2004; Hagmar et al. 2005; Hagmar et al. 2006), providing additional indications of reduced environmental pollution of PCB. Contrary to some of these domestic reports (Norén and Meyronité 2000; Lignell et al. 2004; Hagmar et al. 2006) no such downward trend was observed for HCB in the present study, but the reasons for this discrepancy remained unclear. DDT, the precursor to p,p'-DDE, was banned in Sweden somewhat prior to the ban on PCB and domestic exposure to this substance is likely to have been quite limited since. We are unaware of studies showing a DDT connection to elastic sealants and the somewhat higher GM p,p'-DDE level in the exposed group vs. con-

Table 7 Plasma levels of cytokines (pg/ml) and proportion (%) of samples at or above the limit of quantification (LoQ) in PCB abatement workers (baseline and follow-up) and controls

Cytokine	LoQ pg/ml	Abatement workers				Controls	
		Baseline $n = 36$		Follow up $n = 25$		$n = 33$	
		Range	≥LoQ %	Range	≥LoQ %	Range	≥LoQ %
IL-2	2.6	<2.6–10.7	13.9	<2.6–4.4	8.0	<2.6–29.4	9.1
IL-4	2.6	<2.6	0.0	<2.6	0.0	<2.6–55.0	3.0
IL-6	3.0	<3.0–34.8	30.6	<3.0–22.7	48.0	<3.0–98.9	42.4
IL-10	2.8	<2.8–63.1	30.6	<2.8–87.9	64.0	<2.8–123.2	27.3
TNF-α	2.8	<2.8–19.2	25.0	<2.8–9.0	48.0	<2.8–63.5	24.2
IFN-γ	7.1	<7.1–42.5	25.0	<7.1–51.7	56.0	<7.1–188.3	24.2

trols could thus not be interpreted with available information. There was no *p,p′*-DDE increase at follow-up of the abatement workers, however, suggesting no further exposure.

Due to structural similarities with thyroid hormones, the possible endocrine disrupting potential of PCB has raised a considerable scientific interest and both experimental and wildlife studies have suggested a negative effect of PCB on thyroid function (Porterfield and Hendry 1998; Fisk et al. 2005). By contrast, a literature review of observational studies in humans concluded that there was no convincing evidence of an effect of PCB on thyroid hormone homeostasis (Hagmar 2003).

When analysing highly lipophilic compounds like PCB in human blood samples (serum or plasma) it is customary to standardise the results with regard to the lipid content (Phillips et al. 1989). This procedure might, however, lead to biased estimates of association between exposure and effect, at least in logistic regression (Schisterman et al. 2005), and a preferable method would be to use the wet weight PCB level with serum lipids as covariate in the regression model. This approach was applied by Meeker et al. (2007) in a study of thyroid hormones and serum PCB in men from an infertility clinic. They found significant inverse associations between total T_3 and PCB congeners #118, 138, and 153, both when adjusting exclusively for serum lipids and also after inclusion of age, BMI, current smoking, *p,p*'-DDE, and timing of the venepuncture in the analysis.

In the present study no significant association, positive or negative, was found between the level of PCB and indicators of thyroid function. To check if our results could be an artefact of the choice of statistical model, the data were reanalysed using the Meeker et al. (2007) approach. There were, however, still no significant associations between thyroid hormones and PCB. Moreover, it should be noted that the levels of PCB congeners #138 and 153 were higher in our study than in the study by Meeker et al. (2007), suggesting a genuine lack of association.

Cytokines are a complex group of cell-derived, soluble low molecular polypeptides acting as immune system mediators (Banks 2000; Balkwill 2001). PCB are recognised as immunosuppressive, affecting both humoral (circulating) and cell-mediated components of the immune system (Ahlborg et al. 1992; WHO 1993). Daniel et al. (2001) reported an inverse relationship between PCB #138 in human blood and plasma levels of IL-4 but not with several other cytokines. In the current study, the overall low proportion of quantifiable cytokine results was a limitation in the statistical analysis and any dose-response relationship between PCB levels and IL-2 and IL-4, respectively, could not be evaluated. For the remaining cytokines no statistically significant relation with PCB, positive or negative, was observed although there was a significant change from baseline to follow-up within the exposed group. However, given the contrasting PCB levels in the exposed group and the controls it seems unlikely that this finding could be attributed to PCB. Information on storage effects in cytokine analyses is ambiguous (Banks 2000) but the higher proportion of quantifiable cytokines in the follow-up samples from the exposed group, given overall stable PCB levels ($\sum$19 PCB), would suggest some influence of long-term storage affecting the baseline samples.

It is not common to find a group of small contractors within the construction industry displaying an avant-garde interest in protection of both their workers and the general environment. Accordingly, no inference can be drawn from our results to a wider spectrum of abatement contractors, nor should they be regarded valid prospectively. Increased intensity of exposure, as would be expected from a recent Swedish government ordinance on PCB removal (Ministry of the Environment 2007), a changing workforce and reduced awareness of the risks involved are all factors of concern for the future.

To conclude, this study indicates that PCB abatement workers' PCB uptake can be quite limited given the availability and implementation of an elaborate preventive strategy. The study also suggests that, using clinical standard tests, thyroid metabolism is not affected at the low levels of internal PCB load observed. Finally, no evidence of PCB-associated immunosuppression was noted in a set of cytokine analyses.

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